Case Report

Calcific Tendinitis of the Rectus Femoris

A Rare Cause of Snapping Hip

Luca Pierannunzii MD, Filippo Tramontana MD,
Mauro Gallazzi MD

Received: 19 August 2009 / Accepted: 15 December 2009 / Published online: 7 January 2010
© The Association of Bone and Joint Surgeons® 2010

Abstract

Background Internal snapping hip is a syndrome caused by recurrent subluxation of the iliopsoas tendon. There is little agreement regarding the impinging sites responsible for the jerky motion of the tendon. Thus far, the lesser trochanter, anterior capsule, and iliopectineal eminence are considered the most likely catching sites.

Case Description We report an unusual case in which a calcific tendinitis of the rectus femoris direct head impinged against the overlying iliacus muscle, resulting in a painful coxa saltans. The exclusive involvement of the direct head hid the calcium deposit on standard radiographs, whereas MRI suggested but poorly showed the tendon disease. Dynamic ultrasonography and CT scanning allowed a precise diagnosis and subsequent treatment with CT-guided steroid injection.

Each author certifies that he or she has no commercial associations (eg, consultancies, stock ownership, equity interest, patent/licensing arrangements, etc) that might pose a conflict of interest in connection with the submitted article.

Each author certifies that his or her institution approved the reporting of this case report, that all investigations were conducted in conformity with ethical principles of research, and that informed consent for participation in the study was obtained.

Electronic supplementary material The online version of this article (doi:10.1007/s11999-009-1208-9) contains supplementary material, which is available to authorized users.

L. Pierannunzii (✉)
III Division of Orthopaedics and Trauma, Gaetano Pini
Orthopaedic Institute, Piazza C. Ferrari, 1, Milan, Italy
e-mail: lmpierannunzii@hotmail.com

F. Tramontana, M. Gallazzi
Department of Radiodiagnostics, Gaetano Pini Orthopaedic
Institute, Milan, Italy

Literature Review Calcific tendinitis of the rectus anterior direct head has not been reported as a possible cause of snapping hip; involvement of the direct head in rectus anterior calcific tendinitis was described in one case.

Purposes and Clinical Relevance Our case shows the rectus anterior direct head may be involved in the etiology of coxa saltans. Theoretically any thickening of the tendon might activate the same pathomechanism. Physicians should consider this possible new cause of internal snapping hip when the most common ones have been excluded, especially as it may be managed easily with steroid injection.

Introduction

The snapping hip is a syndrome characterized by recurrent clicks or snaps during active motion of the hip [2, 11]. Occasional painless snaps are felt commonly by patients with normal hips and these should not be regarded as pathologic.

Three kinds of snapping hips are known: an intraarticular variety (attributable to loose bodies, labral lesions, chondromatosis, joint instability, or ruptured ligamentum teres) [2, 5, 10, 20]; an external extraarticular variety (attributable to subluxation of the posterior aspect of the iliotibial band or the anterior border of the gluteus maximus over the greater trochanter) [2, 4, 7, 9]; and an internal extraarticular variety (attributable to subluxation of the iliopsoas tendon) [11].

The pathomechanism of internal extraarticular snapping hip is not well established. Neither jerking of the iliofemoral ligament over the capsule [6] nor the presence of a stenosing tenosynovitis of the iliopsoas tendon [10] has been confirmed. However, although the existence of

iliopsoas tendon subluxation is widely accepted, there is little agreement regarding the possible sites causing the sudden translation. An *in vivo* study by Schaberg et al. [16] suggested the site might be an osseous ridge on the anteromedial side of the lesser trochanter and of the iliopectineal eminence, whereas the role of the anterior inferior iliac spine was suspected but not confirmed.

We present the case of a patient with painful internal snapping hip apparently caused by calcific tendinitis of the rectus anterior direct tendon.

Case Report

The patient was a slender 43-year-old woman with an active lifestyle. Two years previously, she began having occasional right groin pain precipitated by uphill trekking. At first, the discomfort was elicited only by long and steep climbing, but gradually it became more and more frequent, sometimes induced just by climbing stairs and eventually by level walking. One year after the onset, she started feeling a snap in the left hip associated with active extension of the hip from a flexed position. Soon afterward, the snap became audible but never truly painful. The groin pain, however, became more and more disabling, preventing the patient from any kind of activity.

Physical examination showed complete passive ROM, substantially pain-free except in maximum flexion, negative impingement signs, and no tenderness points around the bony landmarks. The resisted flexion of the hip was painful. The snap could be reproduced easily by the patient while she was in a supine position when flexing her hip 90° with straight knee and then lowering it to the examination couch. The snap occurred at approximately 45° flexion and was audible and slightly palpable in the lateral part of the groin. The rotation of the hip did not affect the result of the test. Moving the hip passively from FABER position (flexion-abduction-external rotation) to EAddIR position (extension-adduction-internal rotation) did not elicit any snap, whereas moving it actively produced it only occasionally.

Fig. 1A–B (A) The AP and (B) the frog leg lateral views showed no relevant abnormalities. However, in light of the subsequent examinations, the soft tissues above the right acetabular roof (arrow) appeared slightly more radiopaque than the contralateral ones.

The patient's radiographs at that time (Fig. 1) showed no major osseous or articular abnormalities. A static ultrasound examination revealed no iliopsoas bursitis or tendinopathy but showed a slightly thickened rectus femoris direct head (Fig. 2). Pain by deep passive flexion and essentially unremarkable sonography diverted the attention from any musculotendinous lesions and suggested a labral injury, which also would be consistent with a snapping hip. An arthroMRI therefore was performed but showed no abnormality other than the thickened and edematous direct head of the rectus femoris (Fig. 3). The sonographic examination then was repeated at our

Fig. 2 The first sonograms showed a thickened rectus anterior direct head (arrowheads), clearly visible on the longitudinal plane. A = acetabulum; AIIS = anterior inferior iliac spine; FH = femoral head; IM = iliacus muscle; RA = rectus anterior.

Fig. 3 The axial fast spin echo T1-weighted MR slice through the lower part of the anterior inferior iliac spine shows thickening and an inhomogeneous increased signal of the rectus anterior direct origin (arrow) (TR = 589 ms, TE = 11 ms).

institution, performing static and dynamic studies. A calcification was identified at the origin of the rectus femoris tendon from the anterior inferior iliac spine and the overlying iliacus muscle appeared to be imprinted by the bulge (Fig. 4). The actively controlled extension made the iliacus

muscle belly split abruptly into two parts: the largest one moved medially over the bulge, whereas the most lateral bundle diverged laterally, simultaneously with an audible snap. (Video 1. Supplemental materials are available with the online version of CORR.)

The patient was treated with two steroid injections in the calcific deposit under CT guidance (Fig. 5), the second injection being administered 1 month after the first one for incomplete recovery. Relief of symptoms was fast after the second dose.

The patient returned to her previous activity level 2 months after the second injection, resuming trekking and other sports. At the 1-year followup, the patient remained pain-free. Snaps no longer occurred during ordinary life activities and although they could be reproduced by the patient, they were barely perceptible by the examiner.

Discussion

Calcific tendinitis of the rectus femoris is a rare disease [1] first recognized in 1967 by King and Vanderpool [8]. Causes and pathogenesis are likely similar to other more common calcific tendinitis sites such as the rotator cuff: mechanical stress or vascular impairment (or both) might lead to local hypoxia and to fibrocartilaginous degeneration of the tendon, which would undergo subsequent calcium deposit. The angiogenic response would allow eventual resorption by macrophages and tendon healing [18].

Fig. 4A–C (A) Ultrasound longitudinal static scanning showed a calcification next to the anterior inferior iliac spine imprinting the iliacus muscle. The dynamic transverse scanning during active hip extension from (B) 60° to (C) 20° showed a bundle of iliacus fibers—the ilioinfratrochanteric bundle—diverging laterally over the calcific

rectus origin, while the rest of the muscle belly moves medially. AIIS = anterior inferior iliac spine; Ca = calcification; IITB = ilioinfratrochanteric bundle; IM = iliacus muscle; IMB = iliacus main belly; RA = rectus anterior.

Fig. 5 The axial CT slice through the lower part of the anterior inferior iliac spine shows the calcium deposit clearly and proper placement of the needle for steroid injection.

The usual presentation is characterized by acute onset, severe pain, limitation of hip ROM, and limp [15]. Sometimes the skin over the anterolateral aspect of the proximal thigh is warm [3] and the erythrocyte sedimentation rate and C-reactive protein are above the normal range [14]. In those acute cases, the differential diagnosis with acute arthritis of the hip is challenging. In contrast, our patient did not have an acute phase of inflammation develop, and her symptoms had a slow gradual onset.

Most of the published reports regarding calcific tendinitis of the rectus femoris [3, 8, 14, 15] describe involvement of the indirect head, which surrounds the acetabular roof with a wide insertion largely overlapped with the capsular attachment. This paraarticular location of the calcium deposit might explain the severe limitation of ROM experienced by patients. Pope and Keats [13] reported the case of a patient with direct and indirect tendon calcific tendinitis, but we are unaware of a case of calcific tendinitis of the direct head. The higher distance between the joint and the inflammation site may explain the nonacute presentation in our patient. However, probably for the same reason, none of the other patients with calcific tendinitis of the rectus femoris were reported to complain about snapping hip.

The anatomic site where the iliopsoas catches in internal coxa saltans has been identified variously by authors reporting these cases. Nunziata and Blumenfeld [11], who first recognized this kind of snapping hip in 1951, believed the iliopsoas tendon caught on the iliopectineal eminence. Schaberg et al. [16] also described subluxation of the

iliopsoas tendon over an anteromedial osseous ridge of the lesser trochanter, just above the insertion. Wahl et al. [19] reported the case of an athlete in whom they believed the iliopsoas snapped over an anterior thickening of the hip capsule. In addition to these explanations, the role of the anterior inferior iliac spine has been conjectural so far. The findings in our patient suggest the anterior inferior iliac spine may have a role in the internal snapping hip and impingement location may influence the clinical presentation. The anterior inferior iliac spine is the most lateral structure of the iliopsoas groove and is commonly thought to represent its lateral limit, whereas the medial limit is constituted by the iliopectineal eminence. However, some iliacus muscle fibers actually leave the pelvis laterally to the anterior inferior iliac spine. Those fibers represent the ilioinfratrochanteric bundle, which arises from the notch between the anterior superior iliac spine and the anterior inferior one, and from the upper side of the anterior inferior iliac spine, above the rectus insertion [17]. This bundle is mainly muscular, almost without any tendinous component, and inserts anteriorly and inferiorly to the lesser trochanter.

In our patient, the snap was reproduced only by actively controlled extension of the hip, whereas passive provocative maneuvers were negative. This might depend on the characteristics of the snapping bundle, which, being fully muscular, is loose in absence of contraction. Because the actively controlled extension also determines the rectus femoris contraction, another possible explanation is that such a contraction in flexion raises the proximal tendon (and the included calcification), increasing the conflict against the overlying bundle. Apparently this case of internal snap was not influenced by rotation, although most others are. Although any explanation based on one case is mainly guesswork, we believe the proximal location of the impinging site could reasonably explain such behavior. When the hip rotates internally, the lesser trochanter moves from lateral to medial. The medial translation is much wider if the internal rotation is associated with adduction, as when the hip moves from the FabER position to the EAddIR position. The whole iliopsoas musculotendinous unit is supposed to follow the transverse translation of the lesser trochanter, obviously translating more in the distal part (close to the mobile femoral insertion) than in the proximal one (close to the fixed pelvic and lumbar origins). For this reason, the musculotendinous junction moves over the iliopectineal eminence [12], and the iliopsoas tendon translates even more widely over the capsule [19]. In contrast, the ilioinfratrochanteric bundle could hardly translate over the anterior inferior iliac spine as a consequence of any medial translation of the femur, as the anterior inferior iliac spine is extremely close to the fixed origin of the bundle (interspinous notch) and extremely far from the mobile distal insertion (infratrochanteric area).

Even though the calcific tendinitis of the rectus femoris direct head is extremely rare, other pathologic changes involving this tendon could result in similar thickening, potentially predisposing the development of an internal coxa saltans. Pelsser et al. [12] reported the sonographic findings of 20 patients (36 hips) with snapping hips. In two, the rectus origin was thickened, and in another three, it was painful under the pressure of the sonographic transducer. All of these cases were of the internal variety group. Although Pelsser et al. did not relate those incidental findings to the pathophysiology of the snap, our case might lead to a possible new interpretation of those observations. The somewhat atypical features of our patient might depend on the specific location of the impinging site, more lateral and proximal than in the other most common cases; the findings suggest the anterior inferior iliac spine and rectus femoris direct tendon may participate in the etiology of internal snapping hip.

References

1. Archer BD, Friedman L, Stilgenbauer S, Bressler H. Symptomatic calcific tendinitis at unusual sites. *Can Assoc Radiol J*. 1992; 43:203–207.
2. Binnie JF. Snapping hip. *Ann Surg*. 1913;58:59–66.
3. Braun-Moscovici Y, Schapira D, Nahir AM. Calcific tendinitis of the rectus femoris. *J Clin Rheumatol*. 2006;12:298–300.
4. Brignall CG, Stainsby GD. The snapping hip: treatment by Z-plasty. *J Bone Joint Surg Br*. 1991;73:253–254.
5. Byrd JW, Jones KS. Traumatic rupture of the ligamentum teres as a source of hip pain. *Arthroscopy*. 2004;20:385–391.
6. Howse AJ. Orthopaedists aid ballet. *Clin Orthop Relat Res*. 1972;89:52–63.
7. Jones FW. The anatomy of snapping hip. *J Orthop Surg*. 1920; 2:1–3.
8. King JW, Vanderpool DW. Calcific tendonitis of the rectus femoris. *Am J Orthop*. 1967;9:110–111.
9. Mayer L. Snapping hip. *Surg Gynecol Obstet*. 1919;29:425–429.
10. Micheli LJ. Overuse injuries in children's sports. *Orthop Clin North Am*. 1983;14:337–360.
11. Nunziata A, Blumenfeld I. [Snapping hip; note on a variety] [in Spanish]. *Prensa Med Argent*. 1951;38:1997–2001.
12. Pelsser V, Cardinal E, Hobden R, Aubin B, Lafortune M. Extraarticular snapping hip: sonographic findings. *AJR Am J Roentgenol*. 2001;176:67–73.
13. Pope TL Jr, Keats TE. Case report 773: Calcific tendinitis of the origin of the medial and lateral heads of the rectus femoris muscle and the anterior iliac spine (AIIS). *Skeletal Radiol*. 1992;21:271–272.
14. Rozenbaum M, Slobodin G, Boulman N, Feld J, Avshovich N, Rosner I. Calcific tendonitis of the rectus femoris. (Comment on: *J Clin Rheumatol*. 2006;12:298–300.) *J Clin Rheumatol*. 2008;14:57.
15. Sarkar JS, Haddad FS, Crean SV, Brooks P. Acute calcific tendinitis of the rectus femoris. *J Bone Joint Surg Br*. 1996;78:814–816.
16. Schaberg JA, Harper MC, Allen WC. The snapping hip syndrome. *Am J Sports Med*. 1984;12:361–365.
17. Tatu L, Parratte B, Vuillier F, Diop M, Monnier G. Descriptive anatomy of the femoral portion of the iliopsoas muscle: anatomical basis of anterior snapping of the hip. *Surg Radiol Anat*. 2001;23:371–374.
18. Uhthoff HK, Sarkar K, Maynard JA. Calcifying tendinitis: a new concept of its pathogenesis. *Clin Orthop Relat Res*. 1976; 118:164–168.
19. Wahl CJ, Warren RF, Adler RS, Hannafin JA, Hansen B. Internal coxa saltans (snapping hip) as a result of overtraining: a report of 3 cases in professional athletes with a review of causes and the role of ultrasound in early diagnosis and management. *Am J Sports Med*. 2004;32:1302–1309.
20. Walker J, Rang M. Habitual hip dislocation in a child: another cause of the snapping hip. *Clin Pediatr (Phila)*. 1992;31:562–563.